

Neuropsychological functioning in people with ADHD across the lifespan

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Abstract

ADHD is defined by behavioral characteristics similar to neuropsychological disorders of executive dysfunction. This paper is a literature review of the neurocognitive characteristics of ADHD from early childhood through adulthood. The author addresses the development of the concept of attention and executive function (EF) deficits in ADHD, clinical neuropsychological studies of pre-teenage children, teenagers and adults with ADHD, gender and the role of psychiatric co-morbidity including the relationship of learning disabilities to ADHD, heterogeneity of neuropsychological dysfunctions, experimental neuropsychological studies, the relationship of brain structure to function, psychopharmacology of ADHD, and clinical neuropsychological assessment. The group data clearly supports the hypothesis that executive dysfunctions are correlates of ADHD regardless of gender and age, and these EF deficits are exacerbated by co-morbidity with learning disabilities such as dyslexia. However, there is limited data on children under the age of 5, teenagers from age 13–18, and adults with ADHD over the age of 40. Studies of individual classification of people with ADHD compared to healthy, non-psychiatric controls do not support the use of neuropsychological tests for the clinical diagnosis of ADHD, and indicate that not all persons with ADHD have EF deficits. Some persons with ADHD may have deficits in brain reward systems that are relatively independent of EF impairments. Future research should clarify the multiple sources of ADHD impairments, continue to refine neuropsychological tools optimized for assessment, and incorporate longitudinal, developmental designs to understand ADHD across the lifespan.

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1. Introduction

Although most of our current knowledge about Attention-Deficit/Hyperactivity Disorder (ADHD) developed from clinical observations and research with children, understanding of the disorder in adults is growing rapidly. Children, adolescents, and adults who are diagnosed with ADHD share similar clinical features, comorbidities, and failures in

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major life domains (e.g., academics and work; (Biederman, Mick, & Faraone, 2000; Hechtman, 1992; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993), and possibly brain abnormalities (Seidman, Valera, & Bush, 2004). However, as there is much to learn from different subgroups of persons with ADHD, it is clear that a full understanding of the disorder requires research from a life span perspective — integrating what we know about how it affects both adults and children (Biederman, 1998).

Cross-sectional data suggest that neuropsychological dysfunctions are an important component of the childhood syndrome (Barkley, 1997; Faraone & Biederman, 1998), and a growing literature is suggesting the same for adults (Hervey, Epstein & Curry, 2004). However, are the neuropsychological deficits ubiquitous as suggested by the diagnostic name, “ADHD”? In this article, we review the current state of the literature pertaining to the neuropsychological dysfunctions that are found in children, teenagers and adults with ADHD, focusing particularly on executive functions. Because there is considerable interest in neuropsychological assessment as a clinical tool for persons with ADHD, the nature and validity of clinical neuropsychological assessment for this population will also be addressed.

2. Development of the concept of attention-executive and brain dysfunction in ADHD

ADHD, formerly called “hyperactivity,” “hyperkinesis disorder of childhood,” or “minimal brain dysfunction,” was first described 100 years ago as a childhood disorder found mainly in boys (Still, 1902). Revisions in the diagnostic construct have been made a number of times over the past century (Barkley, 1990). The most important shift occurred in the 1970s, when the concept of attention dysfunction was introduced as the defining feature (Douglas, 1972), and the disorder was re-named accordingly. However, the key symptoms needed for the diagnosis were behavioral descriptions of motor and attentional problems rather than direct cognitive measures of “inattention”. This is an important point I will return to later.

The diagnosis of ADHD is currently made on the basis of developmentally inappropriate symptoms of inattention, impulsivity, and motor restlessness (American Psychiatric Association, 1994, 2000), and three subtypes are recognized: “inattentive,” “hyperactive–impulsive,” and “combined” (reflecting a combination of the other two types). Symptoms must be: 1) observed early in life (before age 7); 2) pervasive across at least two situations; and 3) chronic. The clinical presentation has suggested that ADHD is a neuropsychological disorder, and current theories emphasize the central role of attentional and executive dysfunctions such as disinhibition (Barkley, 1997; Pennington & Ozonoff, 1996).

The similarities between symptoms characterizing ADHD and those of some patients with neurological disorders led to hypotheses that ADHD is a brain disorder affecting the prefrontal cortex (Mattes, 1980). Mattes noted that lesions in the frontal lobe in animals and human neurological patients were often associated with impulsivity, distractibility and hyperactivity (Fuster, 1989). Early support for the “prefrontal” (PFC) or “fronto-striatal” model of ADHD came from the success of stimulant medications, as well as from animal models implicating dopamine pathways (Shaywitz, Klopfer, & Gordon, 1978), that have a strong predilection for PFC. These hypotheses have gradually garnered support from neuroimaging studies. The most consistently replicated brain structural alterations in ADHD in childhood include significantly smaller volumes in the dorsolateral PFC, and regions that project to the PFC including caudate, pallidum, anterior cingulate, and cerebellum (Seidman, Valera, & Makris, 2005). Functional brain abnormalities, mainly described in adults with ADHD, have also consistently implicated the PFC (Bush, Valera, & Seidman, 2005).

As ADHD has increasingly been understood as a developmental brain disorder affecting regions projecting to PFC, neuropsychological theories have tended to emphasize putative dysfunctions of PFC, especially executive dysfunctions (Barkley, 1997; Pennington & Ozonoff, 1996; Tannock, 1998). However, as reviewed by Sergeant, Geurts, Huijbregts, Scheres, and Oosterlaan (2003), there are a number of other models that seek to explain the cognitive and behavioral problems associated with ADHD. These include models invoking the centrality of reward, delay and inhibition (Sonuga-Barke, 1994), disturbances in “cognitive energetics” (Sergeant, Geurts, & Oosterlaan, 2002), and deficits in language (Hinshaw, Carte, Sami, Treuting, & Zupan, 2002; Tannock, 1998). While a model involving executive functions (EFs) remains a strong heuristic for the field, it is also clear that deficits in EFs are common to many psychiatric disorders and certainly are not specific to ADHD (Sergeant et al., 2002).

In considering these theoretical ideas it is also important to recognize that behavioral studies of normal persons and of brain-injured and psychiatric patients have emphasized that attention and EFs are not unitary processes (Mesulam, 1990; Mirsky, Anthony, Duncan, Ahearn, & Kellam, 1991; Posner & Petersen, 1990). Attention refers to a complex set of mental operations that includes focusing on or engaging a target, sustaining the focus over time using vigilance,

encoding stimulus properties, and disengaging and shifting the focus. EFs regulate behavioral output; typically, they involve inhibition and impulse control, working memory, cognitive flexibility, and planning and organization (Denckla, 1989). Working memory (WM) has been defined as the temporary maintenance, manipulation, and storage of information for use in other cognitive operations, such as reasoning (Goldman-Rakic, 1991). It is analogous to a mental “clipboard” that holds information on line for short periods of time, usually seconds.

In addition to the issue of heterogeneity of attention and EFs described above is the notorious difficulty in precise definition of EFs. For example, Sergeant et al. (2002) note that there are “33 definitions of EF” (p. 3). However, most investigators would agree that EFs are self-regulatory functions incorporating the ability to inhibit, shift set, plan, organize, use working memory, problem solve and maintain set for future goals (Pennington & Ozonoff, 1996; Sergeant et al., 2002). Factor analyses have suggested at least four EF factors: response inhibition and execution, working memory, set shifting, and interference control (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). EFs are distinct from other mental functions such as perception or memory. There is, however, considerable overlap with certain components of learning and memory, especially processes involved with encoding and retrieval (Pennington & Ozonoff, 1996).

These attention and EFs have become the focus of current theories concerning the neuropsychological basis of ADHD. Unlike 10 years ago, when cognitive neuropsychological research in ADHD concentrated on “attention deficit” (e.g., vigilance or distractibility), today’s studies examine multiple dysfunctions in the executive processes that control subordinate cognitive processes. Although there is a lack of consensus about the taxonomy of executive processes, there is some agreement that these processes include attention and inhibition, task management, planning, monitoring, and decoding (Barkley, 1997).

One particular executive process, inhibition, has been suggested to be a potential locus of a core deficit in ADHD (Barkley, 1997; Pennington & Ozonoff, 1996). Executive inhibition (as contrasted with motivational inhibition) comes into play in situations that require withholding or suddenly interrupting an ongoing action or thought (suppression of a primary response, as on the Stop-Signal task, (Logan, Schachar, & Tannock, 1997)). It also occurs with the suppression of information that one wishes to ignore, such as an interfering or conflicting stimulus — as on the Stroop test (Nigg, 1999; Stroop, 1935). According to this model, deficient inhibitory control impairs the ability of persons with ADHD to engage other executive control strategies to optimize behavior. Fuster (1989), in particular, has argued that the proficiency of a related executive function, working memory, is dependent on response inhibition and interference control. Deficient inhibitory control can intrude into WM capacity, leading to disruption of WM and interference with planning and organized behavior. While these theories of the neuropsychological basis appear to be conceptually sound, and have face validity with respect to observed behavior, it is important to carefully evaluate the empirical literature to ascertain the support for these ideas. Based on the published literature to this date, it remains unclear whether specific components of EFs are selectively impaired or can account for other deficits.

3. Neuropsychological dysfunctions in childhood ADHD

3.1. Neuropsychological assessment in preschoolers

Although there are more than a hundred studies examining neuropsychological functioning of ADHD in childhood, there are relatively few studies examining such functioning in ADHD preschoolers, ages 3–5 or children just entering school at ages 5–7. Overall, this small body of work is consistent with that observed in older children with the disorder (Valera & Seidman, 2006). Compared to healthy preschoolers, preschoolers with ADHD have been shown to display more inhibitory deficits and be more delay aversive (Dalen, Sonuga-Barke, Hall, & Remington, 2004; Sonuga-Barke, Dalen, Daley, & Remington, 2002), perform more poorly on visual search cancellation tasks (Byrne, Bawden, DeWolfe, & Beattie, 1998; Byrne, DeWolfe, & Bawden, 1998; DeWolfe, Byrne, & Bawden, 1999), visual and/or auditory vigilance tasks (Byrne et al., 1998; DeWolfe et al., 1999) motor control, working memory, and goal directed persistence (Mariani & Barkley, 1997), and tasks of pre-academic skills including tests of memory, reasoning and conceptual development (DuPaul, McGoey, Eckert, & VanBrakle, 2001). The ability of stimulant medications to improve some aspects of attention, observed in older children with ADHD, was demonstrated by Byrne and colleagues (Byrne et al., 1998) in ADHD preschoolers on a visual and auditory vigilance test as well as on a visual-search cancellation test.

There are also a number of neuropsychological studies of ADHD “older preschoolers” ages 5–6 (e.g. Berlin & Bohlin, 2002; Kalff et al., 2002) or 5–7 (e.g. Hanisch, Konrad, Gunther, & Herpertz-Dahlmann, 2004). The results of

these studies are also consistent with EF and inhibitory deficits in ADHD. The older ADHD preschool children performed significantly worse than controls on tasks of visuomotor ability, working memory and attention (Hanisch et al., 2004; Kalff et al., 2002). Some studies also demonstrated that the cognitive dysfunctions were related to levels of hyperactivity and attention (Berlin & Bohlin, 2002; Harper & Ottinger, 1992). Similarities in functioning found in 3–5 and 5–7 year olds in these cross-sectional studies support the hypothesis that neurocognitive deficits are persistent over time across these younger ages (Valera & Seidman, 2006). Nevertheless, this longitudinal hypothesis must be tested explicitly as some researchers have posited developmental changes in neurocognition. For example, Sonuga-Barke (2005) suggests that EF deficits may only emerge some time after impairments in delay aversion, which may be a more fixed characteristic.

3.2. Neuropsychological studies in children ages 6–12 with ADHD

The neuropsychological functioning of elementary school-age ADHD children has been studied extensively since the early 1970s, beginning with the pioneering work by Douglas on vigilance deficits (Douglas, 1972). Numerous clinical studies (at least 100) have compared groups of ADHD children, typically age 6–12, to normal controls and have generally shown group differences (Frazier, Demaree, & Youngstrom, 2004). While the hypothesis of EF impairment has received substantial support, several studies have not found EF deficits in children with ADHD and additional studies have found that children with ADHD perform poorly on some EF tasks but not others (cf, reviews in Barkley, Grodzinsky, & DuPaul, 1992; Pennington & Ozonoff, 1996; Seidman, Doyle, Fried, Valera, Crum, & Matthews, 2004; Sergeant et al., 2002). Moreover, effect sizes are modest (Frazier et al., 2004), usually ranging from 0.4–0.7 using Cohen's *d* (Willcutt et al., 2005).

While not all studies show positive results, in their entirety, these studies indicate that as a group, children with ADHD exhibit sub-average or relatively weak performance on various tasks of vigilance, verbal learning (particularly encoding), working memory, and EFs such as set-shifting, planning and organization, complex problem solving, and response inhibition (Barkley et al., 1992; Fischer, Barkley, Edelbrock, & Smallish, 1990; Grodzinsky & Diamond, 1992; Pennington & Ozonoff, 1996; Seidman et al., 1995; Seidman, Biederman, Faraone, Weber, & Ouellette, 1997; Seidman, Biederman, Monuteaux, Weber, & Faraone, 2000). Deficits on the Stroop color–word test appear to be among the most significant neuropsychological impairments (Barkley et al., 1992). This task, requiring suppression of interference arising from conflicting information (response inhibition) has been shown to be abnormal in large samples of ADHD boys and girls (Seidman, Biederman, Monuteaux, Valera, Doyle, & Faraone, 2005; Seidman, Biederman, Valera, Monuteaux, Doyle, & Faraone, *in press*). It is now clear that girls with ADHD have neuropsychological deficits (Hinshaw et al., 2002; Seidman et al., *in press*) and that the severity and pattern of deficits is largely the same (Seidman et al., 2005). This will be addressed in more detail in the section on “gender differences”.

3.3. Neuropsychological studies in adolescents with ADHD

It is striking and somewhat surprising that despite the plethora of studies of children aged 6–12, there is remarkably little data on teenagers with the disorder. In our own research spanning ages 6–20, we have demonstrated that the executive dysfunctions that characterize the disorder in childhood are also found in teenagers (Seidman et al., 1997; Seidman et al., 2005). These data demonstrate that samples of healthy control children and children with ADHD both improve their performance as they get older, but the deficit between groups remains significant. This persistent picture and the presence of relatively stable structural brain abnormalities in children age 4–18 (Castellanos et al., 2002) support the notion that these abnormalities will be present in adults with ADHD. In the only published longitudinal study of neuropsychological function, Fischer et al. (1990) demonstrated consistent stable impairments from childhood to older teenage years. Before we turn to a review of this adult neuropsychological data, it is important to address the important effects of psychiatric comorbidity, learning disabilities (LD), and gender on the child data, as these may be important confounds or mediating variables.

4. Are neuropsychological abnormalities accounted for by comorbidity?

Persons with ADHD frequently have comorbid antisocial, substance abuse, mood, anxiety, or learning disorders (Biederman et al., 1993). Although spurious comorbidity can occur due to referral and screening artifacts, the review by

Biederman et al. suggested that these artifacts cannot explain the high levels of psychiatric comorbidity. Family studies of comorbidity by the Biederman research team also dispute the notion that artifacts cause comorbidity (Biederman, Faraone, Keenan, & Tsuang, 1991; Faraone, Biederman, Keenan, & Tsuang, 1991). In addition, studies in children (Faraone & Biederman, 1998; Klorman et al., 1999; Seidman et al., 1995; Seidman et al., 2000; Seidman et al., 1995) and adults (Faraone et al., 2000; Seidman, Biederman, Weber, Hatch, & Faraone, 1998) showed that neuropsychological deficits in ADHD remained robust after statistically adjusting for the presence of psychiatric comorbidities. Thus, the existing data suggest neuropsychological abnormalities in ADHD can be demonstrated independent of psychiatric comorbidity. Further research is needed to address whether particular subgroups of persons who have ADHD+other psychiatric disorders (e.g., ADHD+bipolar disorder) are especially or distinctively impaired compared to other subgroups of ADHD children.

4.1. *Learning disabilities*

An additional complex obstacle in identifying the underlying neuropsychology of ADHD pertains to the overlap between ADHD and various kinds of learning disabilities, which by definition are neurocognitive disorders. The literature on ADHD has consistently documented that a substantial minority of children with ADHD also have learning disabilities (LDs), such as reading or arithmetic disability (Cantwell & Satterfield, 1978; Lambert & Sandoval, 1980; Levine, Busch, & Aufseeser, 1982). Rates vary depending on the definition and type of LD, with estimates ranging from 10% to more than 90% (Semrud-Clikeman et al., 1992), although a rate of approximately 30% using both reading and arithmetic as comorbid LDs has been more realistically suggested (Faraone, Biederman, Monuteaux, Doyle, & Seidman, 2001). LDs, when combined with ADHD, have a specific role in school failure (Faraone et al., 2001). Because persons with LDs (without ADHD) can also manifest neuropsychological deficits in attention and in components of memory (Benezra & Douglas, 1988; Denckla, 1991; Whyte, 1994), more work is needed to further evaluate whether neuropsychological deficits in ADHD children with comorbid LDs are due to ADHD or to LD.

Learning disabilities are neuropsychological disorders characterized by specific processing problems. For example, dyslexia (i.e., reading disability) is characterized especially by specific impairments in single word reading, reading fluency, and reading comprehension, usually resulting from deficient phonological processing (Pennington, Groisser, & Welsh, 1993). Although in our work, we documented that EF deficits in ADHD youth remained significant after statistically controlling for the presence of LD (Faraone & Biederman, 1998), questions remain as to the nature of the association between ADHD, LD and executive deficits. For example, in a pilot study of ADHD boys, we found that youth diagnosed with ADHD and LD (ADHD+LD) were significantly worse than those with ADHD without LD (ADHD-LD) on the Rey-Osterrieth Complex Figure organization score (a measure of executive functions) and on rapid naming on the Stroop test (Seidman et al., 1995). However, because our initial results were based on a small sample we could not address specific types of LDs, such as those associated with arithmetic or reading, which were lumped together. An understanding of the role of the specific LDs combined with ADHD is important for clarifying the nature of neuropsychological deficit in ADHD.

In conceptualizing the complex relationship between ADHD and LD in ADHD children, several hypotheses can be formulated. One possibility is that the comorbidity of LD within ADHD represents a qualitatively distinct condition, as suggested by family studies relevant to genetic transmission of the disorders (Faraone et al., 1993). There is also support for this model from a number of studies which demonstrate that reading disability and ADHD are characterized by separate deficits, namely phonological processing deficits in the former and EF deficits in the latter (Pennington et al., 1993; Shaywitz et al., 1995). This leads to the hypothesis that persons with ADHD+LD would not have worse executive function deficits than persons with ADHD without LD.

An alternative hypothesis is that persons with ADHD and comorbid LD have more severe executive deficits than persons with ADHD without LD (Seidman et al., 1995), because of the additive effect of combining two cognitive disorders which both include attentional and memory dysfunctions. There is some support for this hypothesis. A number of studies have compared ADHD children with and without accompanying reading disabilities (RD) on a range of neuropsychological measures. August and Garfinkel (1990) reported that their combined ADHD+RD group performed significantly worse than the ADHD group (which was also impaired relative to normal controls) on a range of measures in the areas of attention, vocabulary, degraded word recognition, and memory for letter sequences. A similar pattern of findings emerged in a study of memory functioning (Katarina, Hall, Wong, & Keys, 1992) in which both ADHD and ADHD+LD groups displayed sub-average recall of sequential/ordered auditory and visual information, with the

comorbid group showing greater difficulty. Tarnowski, Prinz and Nay (1986) found that the ADHD+LD group was significantly worse on perceptual discrimination on a Continuous Performance Test (CPT). In a large recent study, Willcutt et al., (2001) found that ADHD+RD was most impaired on virtually all measures of neuropsychological function compared to persons with ADHD without RD. However, not all studies have found ADHD children with learning problems to be more impaired on measures of memory, attention, and visual-motor functioning (Halperin, Gittelman, Klein, & Rudel, 1984).

Because these findings were obtained in studies composed mainly of preadolescent, elementary school boys (ages 6–12), questions remain regarding the relationship of ADHD and LD in adolescents. Moreover, almost all research attention has been devoted to studying the impact of comorbid reading disability (RD), while the relevance of comorbid arithmetic disability (AD) has been neglected. We could find no published papers addressing the specific role of AD and ADHD on neuropsychological function, nor had prior studies evaluated the neuropsychological consequences associated with combined RD, AD and ADHD. These results strongly suggested that additional analyses of specific LDs associated with ADHD is important.

An additional issue has to do with the definition of LD. There are no strategies for the definition of LD accepted by all investigators, and definitions vary, at least in part, in relation to educational criteria, state regulations, and neuroscientific models (Fletcher, Francis, Rourke, Shaywitz, & Shaywitz, 1992; Fletcher et al., 1994). In our early work on LD and ADHD, (Faraone & Biederman, 1998; Faraone et al., 1993; Faraone et al., 2001; Seidman et al., 1995; Seidman et al., 2000; Seidman et al., 1998; Semrud-Clikeman et al., 1992) we have used a regression-based approach correcting for the correlation of IQ and achievement as recommended by Reynolds (Reynolds, 1984) and Frick (Frick et al., 1991) to define LDs. We recognize that there is evidence suggesting that low achievement is an equally valid method of classifying persons with LD (Fletcher et al., 1992; Fletcher et al., 1994) and that alternative methods of classification need to be compared. Thus, in the studies we report below, we combined these methods.

We tested a number of hypotheses regarding the relationship of ADHD and LD to neuropsychological dysfunctions (Seidman, Biederman, Monuteaux, Doyle, & Faraone, 2001). We studied the effect of comorbid reading or arithmetic learning disabilities (LDs) on neuropsychological function in ADHD. Participants were 148 males diagnosed with DSM-III-R ADHD, with ($N=69$) and without ($N=79$) LD, and 127 non-ADHD, non-LD male controls of similar age (range 9–22). LD was defined by a combined regression based+low achievement classification. Analyses adjusted for the effect of psychiatric comorbidity, age and socioeconomic status on neuropsychological function. Children who had ADHD+LD were significantly more impaired on both executive and non-executive functions than ADHD children without LD. Neuropsychological performance was most impaired in ADHD with combined Arithmetic and Reading Disability. These data indicate that comorbid LD, especially Arithmetic Disability, significantly increases the severity of executive function impairment in ADHD.

We have recently replicated most of these results in a large sample of girls with ADHD, in which the ADHD+LD subgroup, again using a combined regression based+low IQ approach, demonstrates significantly more neuropsychological impairment than the ADHD–LD subgroup (Seidman et al., in press). Our results lead to a number of conclusions. First, a substantial component of neuropsychological deficit in ADHD is explained by LD comorbidity. Second, having LD+ADHD predisposes to a particularly severe form of executive dysfunction that requires careful attention in clinical assessment and interventions for both girls and boys.

5. Heterogeneity of ADHD and neuropsychological abnormalities

We have already addressed the fact that ADHD is a heterogeneous clinical disorder with substantial psychiatric and cognitive comorbidity. However, we have not yet investigated the issue of whether neuropsychological deficits are present in all or most individuals with ADHD, or whether the observed group differences can easily be applied to the individual case. To some, this may seem confusing: if ADHD is named “attention deficit (hyperactivity) disorder”, shouldn’t all cases with the disorder have “attention” deficits? This would be true if the attention-executive functions measured by psychological laboratory tests are synonymous with the behaviors and symptoms that make up the diagnostic criteria. It’s possible that there is substantial “method variance” (differing methods potentially yielding different results) in the different measures of assessing “attention”, and that attention-executive function neuropsychological deficits will not characterize all persons with the diagnosis.

In fact, the results suggest that neuropsychological and behavioral assessments of ADHD executive functions may not always be measuring the same thing. Variability across studies has been noted by comprehensive reviews of the

literature (Barkley et al., 1992; Pennington & Ozonoff, 1996; Sergeant et al., 2002). For example, Sergeant et al. (2003) found that many but not all studies found Stroop interference deficits, Wisconsin Card Sorting Test (WCST) impairments and reduced verbal fluency using letters in ADHD samples. Although in their 1992 review, Barkley et al., (1992) speculated that the variability could be due to methodological differences and small sample sizes, the fact that heterogeneity continues to emerge across large-scale studies as well as within studies suggests that there is true variability of EF. Performance differences within ADHD samples have been documented by studies that have examined whether various measures of EFs could be used as diagnostic tools for ADHD. These studies have examined male (Doyle, Biederman, Seidman, Weber, & Faraone, 2000; Grodzinsky & Barkley, 1999; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005) and female (Hinshaw et al., 2002) youth as well as adults (Lovejoy et al., 1999) and found that most measures of EFs have good positive predictive power for ADHD (characterized by adequate sensitivity) but poor negative predictive power (poor specificity). That is, abnormal scores on measures of EFs are generally predictive of the diagnosis; however, normal scores cannot rule out the diagnosis. This pattern is due to the fact that not every subject is impaired on every test and that some individuals with ADHD perform within the normal range on all or most measures. The research suggests that approximately 30–50% of persons with ADHD can be considered to be neuropsychologically abnormal in the context of approximately 5–10% abnormality in controls (Doyle et al., 2000; Nigg et al., 2005). This surprising picture may characterize true variability in neuropsychological dysfunctions in ADHD, or some methodological aspects of the tests or testing situation that limits their sensitivity (i.e., the test situation enhances structure in a way that minimizes EF deficits). While refinements in testing methods should proceed, at this point in time neuropsychological tests *cannot* be used for diagnosis.

Another area of investigation evaluating heterogeneity of EF in ADHD is using DSM IV subtypes — Combined, Hyperactive or Inattentive. This literature is relatively sparse and the few studies comparing these subtypes have yielded equivocal findings. For example, Faraone, Biederman, Weber, and Russell (1998) did not find cognitive differences between subtypes on academic measures or IQ estimates. Whereas, Klorman et al., (1999) found that combined and hyperactive children performed more poorly than inattentive children on the Tower of Hanoi (but not on the WCST), Houghton et al. (1999) did not find significant differences between combined and inattentive subtypes on the WCST, Trails, Stroop, Tower of London or Matching Familiar Figures. Nigg, Blaskey, Huang-Pollock and Rappley (2002) reported that on most of the domains they studied, the combined and inattentive subtypes did not differ. In boys with ADHD, they reported significantly more response inhibition deficits in the combined than inattentive subtype. At this point, the literature suggests more similarities than differences in EF in ADHD subtypes, and the more relevant distinction appears to be whether a person with ADHD has an EF deficit or not. Similarly, while “sluggish tempo” has been studied and considered by some to be a subgroup within the inattentive subtype (McBurnett, Pfiffner, & Frick, 2001), it is not clear yet whether it confers a specific association with EF.

6. Effects of gender on neuropsychological abnormalities in ADHD

Although ADHD affects both genders, most of the research literature, including studies evaluating neuropsychological functioning, has been devoted to males (Berry, Shaywitz, & Shaywitz, 1985; Gaub & Carlson, 1997). Gaub and Carlson’s review (Gaub & Carlson, 1997) indicated that few studies included sufficient numbers of female subjects to warrant gender-based conclusions. Nevertheless, there are data supporting the presence of a valid syndrome of ADHD in girls (Hinshaw et al., 2002). Recent work by our group (Biederman et al., 2002), reporting on one of the largest datasets to date on girls with ADHD, identified more similarities than differences in the core features of ADHD with a few notable exceptions; girls were more likely than boys to have a somewhat higher rate of predominantly inattentive type of ADHD (although the combined type was the leading type in both genders), a lesser likelihood to have a LD, a lesser likelihood to manifest problems in school or in their spare time, and a lower risk for co-morbid conduct disorder and oppositional defiant disorder (Biederman et al., 2002). We also demonstrated that the familial transmission of ADHD and comorbid disorders was similar in boys and girls (Faraone et al., 2001; Faraone et al., 2000).

A review of early research suggested that ADHD girls are more neuropsychologically impaired than ADHD boys (Gaub & Carlson, 1997). This observation, while receiving some support for measures of intelligence, does not generalize to EFs, which only partially overlap with measures of intelligence (Pennington & Ozonoff, 1996). In fact, most studies suggest that, though there are neuropsychological impairments in ADHD girls compared to control girls, there are no differences between ADHD girls and ADHD boys on EFs. For example, DeHaas (DeHaas, 1986) showed that both girls and boys with ADHD scored significantly below controls on digit span and all sub-tests of the Stroop.

However, there were no significant differences between ADHD girls and ADHD boys. Similarly, Houghton et al. (1999) found differences between ADHD girls and controls on the Stroop and WCST, but failed to find differences between ADHD girls and boys. Several studies (Arcia & Conners 1998; Breen, 1989; Horn, Wagner, & Ialongo, 1989; Klorman et al., 1999; Schuerholz, Singer, & Denckla, 1998; Sharp et al., 1999) observed putative EF deficits but did not use female controls or did not report within-sex group differences in females. Castellanos et al. (2000) demonstrated that ADHD girls performed more poorly than healthy controls on delayed response and go–no-go oculomotor tasks, consistent with EF impairments that have been noted in boys, but did not examine gender differences. Nigg (1999), using a stop-signal task measuring inhibition, showed that ADHD girls were slower to respond than were controls.

Only two studies found significant gender differences between ADHD boys and girls on attention and executive tasks, and these were minor effects in the broad context of the large number of statistical tests used. Rucklidge and Tannock (2001) found that girls and boys with ADHD (age 13–16) were both impaired in processing speed compared to normal teenagers, but the ADHD boys were slower in processing speed than ADHD girls. Newcorn et al. (2001) found that ADHD girls made significantly fewer CPT impulsivity errors than did ADHD boys, although no normal controls were studied. Many statistical tests did not yield differences.

This literature review suggests there are EF impairments in girls with ADHD but there is limited data about gender differences on measures of EF. Moreover, methodological limitations impede conclusive interpretations. These include: 1) small sample sizes that do not provide enough power to be conclusive; 2) failure to routinely include a substantial group of male and female controls to address normal sex differences (Arnold, 1996); 3) relatively limited sets of EF measures that may not enable an evaluation of an appropriate range of measurement; and 4) failure to control for the common psychiatric comorbidities and learning disabilities.

Two large scale studies have now been carried out that address these issues and confirm that girls with ADHD have neuropsychological impairments (Hinshaw et al., 2002; Seidman et al., *in press*) and that boys and girls with the disorder have similar deficits. In our study, we evaluated if girls with ADHD have EF impairments compared to healthy controls, whether their neuropsychological features are similar to those found in boys, and whether there are systematic sex differences (Seidman et al., 2005). We concluded that girls with ADHD have significant impairments in EFs and that neuropsychological measures of these functions are equally impaired in girls compared to boys with ADHD in pre-teen and teenage subgroups. As with boys, the observed neuropsychological deficits were not accounted for by psychiatric comorbidity or LD. Neuropsychological deficits were most impaired in girls with ADHD+LD and those not taking stimulant medications.

7. Neuropsychological dysfunctions in adult ADHD

Over the past decade, research on clinical neuropsychological dysfunctions in adult ADHD has intensified, and the evidence for such deficits in adults with ADHD is mounting. Recently, a meta-analysis was conducted of neuropsychological deficits in adults with ADHD (Hervey, Epstein, & Curry, 2004). They included only samples with persons 18 years and older, and with a control group. They reviewed 33 published studies and found that neuropsychological deficits are largely consistent with those described in children. Impairments were relatively consistently observed in attention, behavioral inhibition, and memory. Similar results were derived from a qualitative review (Woods, Lovejoy, Stuuts, Ball, & Fals-Stewart, 2002). The adult literature is similar to the child literature described earlier in terms of tests used and other methodological features. Most research is based on the criteria described in the DSM (Diagnostic Statistical Manual; American Psychiatric Association, 1994) after a diagnostic interview. Because ADHD is a developmental disorder considered to begin by age 7, ADHD symptoms in adults are typically assessed with a retrospective report. Hervey et al. (2004), point out that not all studies report the age of the subjects. According to their review, for those studies reporting this important variable, the range is between 19 and 41, with a mean age of approximately 32 years. Thus, the literature is based largely on young adults.

Neuropsychologists have literally hundreds of tests to choose from in composing an assessment battery. In the review of the adult literature for this paper, more than 70 tests used to compare ADHD adults and controls were identified. However, many of these tests were used in only one or two studies and their sensitivity cannot yet be determined. The discussion below will focus on the five tests that most consistently differentiated persons with ADHD from controls and were used in at least seven studies: versions of the CPT, the Stroop, Trail Making, Verbal Fluency (“FAS”) and the Wechsler Adult Intelligence Scale (WAIS). In addition, we will also address the WCST, which consistently yielded non-significant results in adults.

The CPT, originally published by Rosvold, Mirsky, Sarason, Bransome, and Beck (1956) is actually an experimental method that can be endlessly varied to examine different components affecting vigilance and sustained attention (Mesulam, 1990; Mirsky et al., 1991; Posner & Petersen, 1990). Thus, there are many different versions of the CPT in clinical use, some of which are published commercially. Moreover, like many neuropsychological tests, the CPT produces multiple dependent variables, which can reflect different components of mental abilities (i.e., reaction time, errors of commission, errors of omission, etc.). It is beyond the scope of this paper to address all of these variables systematically, but reference will be made to particular variables as needed. We found that different versions of the CPT significantly differentiated adults with ADHD in 13 studies (78%) whereas four studies yielded negative results. The Conners CPT was significantly different in five studies (Barkley, Murphy, & Kwasnik, 1996; Epstein, Conners, Sitarenios, & Erhardt, 1998; Epstein, Johnson, Varia, & Conners, 2001; Murphy, Barkley, & Bush, 2001; Walker, Shores, Trollor, Lee, & Sachdev, 2000) and negative in two (Kovner, Budman, Frank, Sison, Lesser, & Halperin, 1998; Roy-Byrne et al., 1997). The Gordon Diagnostic System (Gordon, McClure, & Aylward, 1989) was significantly different in the three studies in which it was used (Holdnack, Moberg, Arnold, Gur, & Gur, 1995; Johnson, Epstein, Waid, Latham, Voronin, & Anton, 2001; Rapport, Van Voorhis, Tzelepis, & Friedman, 2001). A number of other “home-grown” visual CPTs have successfully discriminated ADHD subjects from controls (Buchsbaum et al., 1985; Gansler et al., 1998; Gualtieri, Ondrusek, & Finley, 1985; Klee, Garfinkel, & Beauchesne, 1986), and a few studies have been negative (Kovner et al., 1998; Weyandt, Rice, Linterman, Mitzlaff, & Emert, 1998), the latter with the Tests of Variable Attention (TOVA). Our study discriminated the groups with a “home-grown” auditory CPT (Seidman et al., 1998). According to Hervey et al. (2004), the effect size (Cohen, 1988) discrimination between persons with ADHD and controls was generally in the moderate ($d=0.50$) to large range (0.75). Commission and omission errors had roughly the same discriminating power. Our results on a relatively simple “X” version of the auditory CPT are consistent with a moderate effect (Seidman et al., 2000).

The Stroop task, generally considered a task of inhibition (at least for the color–word conflict condition) has been used in 15 studies, 11 of which have shown significant discrimination (73%). Most of the Stroop studies have used Golden’s version (Golden, 1978), and eight of twelve of these were significantly impaired in ADHD (Buchsbaum et al., 1985; Corbett & Stanczak, 1999; Dinn, Robbins, & Harris, 2001; Johnson et al., 2001; Murphy et al., 2001; Rapport et al., 2001; Taylor & Miller, 1997; Walker et al., 2000). Four studies using the Golden version had negative results (Downey, Stelson, Pomerleau, & Giordiani, 1997; Sandson, Bachna, & Morin, 2000; Seidman et al., 1998; Silverstein, Como, Palumbo, West, & Osborn, 1995). Two other versions of the Stroop task were significantly impaired in ADHD adults (Hopkins, Perlman, Hechtman, & Weiss, 1978; Lovejoy et al., 1999; Woods et al., 2002). Hervey et al. (2004) report a medium effect size of approximately 0.45 for the color–word test, which is the most discriminating sub-test. Thus, while it is a consistent finding in both adults and children, the effect is modest.

The Trail Making test (Reitan, 1958), a measure involving connecting circles on a page, has two versions: Trails A essentially measures some combination of visual search and perceptual motor speed. Trail Making B adds an executive component, shifting set. Hervey et al. (2004) report a moderate effect size for Trails A (roughly 0.50–0.55), and a slightly larger effect size for Trails B (0.68). We find that seven out of ten studies (70%) show that adults with ADHD perform significantly worse than controls (Gansler et al., 1998; Holdnack et al., 1995; Johnson et al., 2001; Lovejoy et al., 1999; Rapport et al., 2001; Taylor & Miller, 1997; Woods et al., 2002). Three studies found no effect (Sandson et al., 2000; Silverstein et al., 1995; Walker et al., 2000). Thus, while Trails B seems to be a bit more sensitive to the deficits associated with ADHD, the improvement in sensitivity is small, suggesting that processing speed, which is common to both Trails “A” and “B” is an important component of the deficit.

The Controlled Word Association Test (COWAT; Benton, Hamsher, & Sivan, 1983) measures verbal fluency in response to single letters (“FAS”), which taps into phonological associations, and category fluency (“name all the animals you can”). This measure seems to combine rapid access to the lexicon, persistence, and processing speed. Seven of eight studies (87%) demonstrate impairment in adults with ADHD (Dinn et al., 2001; Johnson et al., 2001; Lovejoy et al., 1999; Murphy et al., 2001; Rapport et al., 2001; Sandson et al., 2000; Woods et al., 2002) with only one negative result (Barkley et al., 1996). According to Hervey et al. (2004) most studies have used COWAT total words and the effect size is moderate (0.60).

There is a long tradition of using sub-tests or factors from Wechsler intelligence scales for children and adults in the assessment of ADHD. Most of the published adult research literature is based on the WAIS-R (Wechsler, 1981), which is less sophisticated in its factor structure than the more recently re-normed Wechsler Adult Intelligence Scale-III (WAIS-III; Wechsler, 1997). Clearly, measures of perceptual motor output speed as assessed by the digit symbol coding

test are commonly impaired in adults with ADHD (Buchsbaum et al., 1985; Downey et al., 1997; Gualtieri et al., 1985; Holdnack et al., 1995; Silverstein et al., 1995). Digit Symbol has a moderate to large effect size of 0.62 (Hervey et al., 2004) and along with the Arithmetic sub-test, which taps into working memory, is the most discriminating sub-test. Estimated Full Scale IQ, which is typically calculated as a matching variable between ADHD and control groups (and typically measured with a short form of the Wechsler tests excluding measures of attention) is usually lower in ADHD than controls with a more modest effect size of 0.39 (Hervey et al., 2004). Thus, general cognitive impairments account for a significant proportion of the variance of cognitive deficit in ADHD adults, similar to that observed in children (Faraone & Biederman, 1998).

The issue arises as to whether EF, attention and learning deficits observed in ADHD are a function of IQ. In previous research, many studies have demonstrated that IQ is associated with ADHD (meta-analysis by Frazier et al., 2004). However, caution must be exercised in matching for IQ (Meehl, 1970), because controlling for IQ removes a portion of the variance directly attributable to the independent variable of interest (e.g., ADHD). Moreover, results should be viewed cautiously, when, as in the case of IQ, the covariate shares variance with both independent and dependent variables (Miller & Chapman, 2001). Studies that used IQ as a covariate to determine if neuropsychological deficits were present over-and-above IQ differences tend to show differences although there is some attenuation of results, supporting the conclusion that some of the EF impairments in ADHD go beyond intellectual deficits assessed by IQ (meta-analysis by Frazier et al., 2004).

Overall, we found significant impairments in 13 of 18 (72%) studies using the WAIS (Buchsbaum et al., 1985), the WAIS-R (11 positive reports: (Barkley et al., 1996; Biederman et al., 1993; Holdnack et al., 1995; Klee et al., 1986; Kovner et al., 1998; Lovejoy et al., 1999; Matochik, Rumsey, Zametkin, Hamburger, & Cohen, 1996; Silverstein et al., 1995; Taylor & Miller, 1997; Walker et al., 2000; Woods et al., 2002) and 4 negative results: (Gansler et al., 1998; Rapport et al., 2001; Sandson et al., 2000; Seidman et al., 1998), and the WAIS-III (Murphy et al., 2001). Conceptually, the Working Memory and Processing Speed indexes of the WAIS-III are most likely to be impaired in persons with ADHD and we expect more studies of these indexes to emerge shortly with the increased use of the WAIS-III.

It is of interest that the WCST is quite ineffective in distinguishing adults with ADHD from controls. The WCST has long been considered, in the general neuropsychological literature, to be one of the premiere measures of executive functions (Seidman & Bruder, 2003). The first factor of the WCST, Perseveration (Koren et al., 1998) is a classic measure of dorsolateral prefrontal cortical function. However, in our review of studies with adults with ADHD, only one study showed a positive result (Taylor & Miller, 1997), and seven did not (Gansler et al., 1998; Holdnack et al., 1995; Johnson et al., 2001; Matochik et al., 1996; Rapport et al., 2001; Seidman et al., 1998; Weyandt et al., 1998). Consistent with our summary, Hervey et al. (2004) report trivial effect sizes of 0.02 for categories completed and 0.12 for perseverative errors. Given that the WCST has adequate discriminability in children with ADHD two possibilities can be considered to explain this difference in sensitivity: 1) The test is simply too easy for adults because it has a “low ceiling”. That is, normal children can achieve approximately adult levels by ages 10–12 (Heaton, Chelune, Talley, Kay, & Curtiss, 1993; Mannuzza et al., 1993); 2) Adults with ADHD improve on this task relative to controls from childhood to adulthood. Although this latter possibility can only be answered definitively by a longitudinal study, we favor the former interpretation. The WCST was developed in an era (Milner, 1963) when the major focus was on evaluating adults with significant brain damage, such as structural damage to prefrontal cortex as a result of tumors etc. (Seidman & Toomey, 1999). It simply was not developed to assess persons with subtler neurodevelopmental disorders. Other tasks of problem solving and abstract reasoning that have higher performance ceilings and have better psychometric features (such as various Tower tasks, or more difficult sorting tasks) may turn out to be effective discriminators of adults with ADHD from controls.

It is important to note that the tests described above are well-studied clinical instruments and may be less (or possibly more) sensitive to underlying cognitive impairments in adults with ADHD than are a newer generation of information processing and experimental neuropsychological tests (Seidman & Bruder, 2003). Given the hypothesized deficits in attention and EF, especially inhibition, a number of tests hold considerable promise, but too little research has been published yet to summarize the results. The tests that may turn out to be very useful include measures of inhibition such as the Stop-Signal test (Logan et al., 1997) and the multi-source interference test (Bush, Shin, Holmes, Rosen, & Vogt, 2003), tests of working memory such as the Paced Auditory Serial Addition Test (Gronwall, 1977) and the Delayed Oculomotor Response Task (Castellanos et al., 2000), problem solving tasks such as Tower tasks, and tests of timing or temporal discrimination.

In summary, the neuropsychological difficulties found in adults with ADHD (in subjects up to age 40 or so) appear to be qualitatively similar to those seen in children with the disorder; thus, they support the notion of syndromatic continuity.

Nevertheless, additional research is needed because not all studies demonstrate impairment of the same tasks or functions, nor do all studies control for the various confounds (e.g., psychiatric comorbidities) associated with the disorder. Moreover, there is a paucity of longitudinal neuropsychological research from childhood into adulthood and this type of design is necessary to determine the full extent of neuropsychological continuity. Finally, newer and more appropriate tests of the hypothesized cognitive functions underlying ADHD need to be tested in multiple, carefully designed studies.

8. Experimental measures of neuropsychological dysfunctions

In contrast to the large number of clinical neuropsychological studies, paradigms from experimental psychology and cognitive neuroscience have been employed more sparingly and yet they offer much potential to illuminate basic, elemental processes impaired in ADHD. These will be reported selectively to illustrate some approaches to this area. For example, experimental investigations of response inhibition or interference control (Bush et al., 1998) have demonstrated excessive sensitivity to processing irrelevant information in Stroop paradigms (Carter, Krener, Chaderjian, Northcutt, & Wolfe, 1995a). Asymmetrical performance deficits on a covert orienting task implicating abnormal right hemisphere processing (Carter, Krener, Chaderjian, Northcutt, & Wolfe, 1995b) have also been observed in ADHD. These types of paradigms allow a possible link to brain laterality or neural processes.

Both behavioral and empirical evidence have provided support for the hypothesis that ADHD individuals have timing or temporal processing deficits. This approach to the study of ADHD has quickened recently as empirical studies of ADHD children have shown impairments in both motor and perceptual timing similar to those observed in cerebellar lesions, and this is important because cerebellar volume is altered in ADHD (Castellanos et al., 2002). Motor timing has typically been assessed by simple finger tapping tasks, with or without a pacing mechanism. Perceptual or temporal processing has been assessed in the form of duration reproduction (e.g., Barkley, Koplowitz, Anderson, & McMurray, 1997; Barkley, Murphy, & Bush, 2001; Meaux & Chelonis, 2003), duration discrimination (e.g., Toplack, Rucklidge, Hetherington, John, & Tannock, 2003), verbal time estimation (e.g., Smith, Taylor, Rogers, Newman, & Rubia, 2002), tapping (e.g., Rubia, Noorloos, Smith, Gunning, & Sergeant, 2003), and anticipation (Rubia et al., 2003) tasks. The results consistently show deficits in either accuracy or variability of performance for ADHD children compared to controls. Though virtually all of the studies cited above have assessed children and adolescents, two studies that have examined young adults also found poorer performance in the ADHD group (Barkley et al., 2001; Seri, Kofman, & Shay, 2001). Additional research using these types of paradigms, in conjunction with commonly used clinical neuropsychological tasks would elucidate their selective contribution to neuropsychological impairment in ADHD.

9. Relating brain structure, function, and neuropsychological dysfunctions

The analysis of attention and EFs into subcomponents, and the mapping of attentional functions onto different brain regions, support the proposition that response inhibition and other executive deficits in ADHD will be associated with structural and functional brain abnormalities in specific regions. However, there is currently limited ADHD research in this area. In children, Casey et al. (1997) found that performance on three response inhibition tasks correlated only with those anatomical measures of fronto-striatal circuitry observed to be abnormal in ADHD (i.e., the prefrontal cortex, caudate, and globus pallidus, but not the putamen). The significant correlations between task performance and anatomical measures of the prefrontal cortex and caudate nuclei were predominantly in the right hemisphere, supporting the role of right fronto-striatal circuitry in response to inhibition and ADHD. Semrud-Clikeman et al. (2000) also studying children, found a significant relationship between reversed caudate asymmetry and measures of inhibition (as measured by the Stroop) and externalizing behavior. Three functional imaging studies of adults, provide evidence that the anterior cingulate (Bush et al., 1999), prefrontal cortex (Schweitzer, Faber, Grafton, Tune, Hoffman, & Kilts, 2000) and cerebellum (Valera, Faraone, Biederman, Poldrack, & Seidman, 2005) are dysfunctional when performing response inhibition and working memory tasks.

There is some limited evidence from studies of ADHD children that executive dysfunctions associated with ADHD are correlated with brain volume abnormalities. Poorer performance on sustained attention tasks was related to smaller volume of the right hemispheric white matter (Semrud-Clikeman et al., 2000). Castellanos et al. (1996) found that Full Scale IQ score correlated significantly with total brain volume and with left and right prefrontal regions. Using the same sample, researchers found in a different report that full scale IQ correlated with cerebellar volumes in ADHD (Berquin et al., 1998). The area of the rostral body of the corpus callosum was significantly correlated with scores on

the impulsivity/hyperactivity scale of the Conners questionnaire (Giedd et al., 1994). These studies were conducted on boys with ADHD. The only study of girls demonstrated that the pallidum, caudate, and prefrontal brain volumes correlated significantly with ratings of ADHD severity and cognitive performance (Castellanos et al., 2001). The extant data, while limited, suggest that impairment on neuropsychological measures of executive dysfunction are associated with abnormal brain structures in ADHD.

10. Psychopharmacology of cognitive deficits in ADHD

The mainstay of clinical intervention with children who have ADHD for the past 50 years has been the stimulants (APA, 2000; Spencer et al., 1996). There is a substantial body of evidence that certain cognitive deficits, such as processing speed, reaction time, vigilance, distractibility, and short-term memory, have been shown to improve with stimulant treatment (Berman, Douglas, & Barr, 1999; Loiser, McGrath, & Klein, 1996; Musten, Firestone, Pisterman, Bennett, & Mercer, 1997; Rapoport, Buchsbaum, & Weingartner, 1980). For example, on the CPT, stimulants improve both omission and commission error types. Other cognitive functions, including executive functions, have been studied less thoroughly and appear to have a smaller magnitude of benefit thus far (Kempton, Vance, Maruff, Luk, Costin, & Pantelis, 1999). However, as more attention is paid to the executive functions, some aspects may be amenable to stimulant or other psychopharmacological treatments and other components may not respond. For example, methylphenidate improves response inhibition on the stop-signal task in children (Tannock, Schachar, Carr, Chajczek, & Logan, 1989) and adults (Aron, Dowson, Sahakian, & Robbins, 2003). Stimulant medications primarily target dopaminergic and noradrenergic pathways. In particular, the mesocortical dopamine system may be largely affected and altered in ADHD (Ernst et al., 1999; Heilman, Voeller, & Nadeau, 1991). Newer non-stimulant treatments such as atomoxetine have shown promise in treating measures of response inhibition on the Stroop, (Spencer et al., 1998). Ultimately, neuropsychological research aims to link the cognitive dysfunctions of ADHD and their underlying neurochemistry in order to further improve cognitive function with pharmacological approaches.

11. Clinical neuropsychological assessment

Although groups of children and adults with ADHD on average perform worse than normal controls on tests of attention and executive function, accumulating data suggest that not all children with ADHD suffer from neuropsychological dysfunction. Therefore, the diagnosis of ADHD should not be ruled in or out using individual neuropsychological test scores or neuropsychological batteries. Using a battery of conventional neuropsychological tests (i.e., Stroop, WCST, CPT, etc), Doyle et al. (2000) demonstrated neuropsychological impairment (defined as impairment on two or more tests, one standard deviation below the control mean) in roughly 35–40% of boys with ADHD ($n = 113$), compared with approximately 10% of normal boys ($n = 103$). However, the majority of children with ADHD did not perform poorly on all 7 tests of cognitive function, and children with ADHD exhibited variable deficits on tests of attention and executive function. These data are consistent with a number of other studies attempting to assess the ability of neuropsychological test performances to classify children (Grodzinsky & Barkley, 1999; Hinshaw et al., 2002) or adults (Lovejoy et al., 1999) with ADHD. Moreover, the results are no more promising in studies comparing neuropsychological profiles in persons with ADHD with people who have a variety of other neuropsychiatric disorders (Sergeant et al., 2002). These results suggest that the ability to use neuropsychological tests to specifically diagnose ADHD is weak.

However, neuropsychological testing may be useful for purposes other than diagnosis. The neuropsychological examination typically has three general aims (Seidman & Bruder, 2003; Seidman & Toomey, 1999): 1) identification of neuropsychological dysfunction leading to inferences regarding the presence, type, and etiology of brain dysfunction; 2) comprehensive assessment of cognitive, perceptual, and motor strengths and weaknesses as a guide for treatment; 3) assessment of the level of performance over a broad range, for both initial evaluation and measurement of change over time. It is only the first aim that is significantly limited by the moderate level of sensitivity and specificity of neuropsychological tests for ADHD. The latter two aims are often very applicable to persons with ADHD. Moreover identifying the subgroup of ADHD persons with EF impairments is important because these deficits are related to real world impairments (Biederman et al., 2004).

In assessment of ADHD, the neuropsychologist often begins with a series of short, focused tests and adds other tests of mental functions that need to be addressed in more detail. In ADHD the diagnosis is made by DSM symptoms and

history. Yet the clinician may want to address the degree of formal attention or executive dysfunction that is present. This typically leads the examiner to evaluate vigilance (using a CPT), response inhibition or interference (such as by use of the Stroop or stop-signal test) and organizational skills (such as by use of the Rey–Osterrieth Complex Figure (Seidman et al., 1995; Teknos, Bernstein, & Seidman, 2003)). Because learning disabilities such as reading or arithmetic disability overlap commonly with ADHD and contribute to executive dysfunctions (Seidman et al., 2001) and long-term school outcome (Faraone et al., 2001), it is often important to add a number of tests to address the presence of co-occurring learning disability. Thus, the examiner may bring in measures of phonological processing (for assessment of dyslexia, Willcutt et al., 2001) and/or measures of mathematical and spatial ability, to assess non-verbal learning disability (Seidman & Toomey, 1999). The individual case assessment requires a flexible, hypothesis testing approach in which different tests are used with different patients, and no single test or battery can be recommended for all testing purposes at this point in time.

A population of increasing interest is the older teenager and young adult with ADHD, many of whom are in college, graduate school or are employed. Many of these patients no longer live at home but are at the border of financial and psychological independence from their families. For such patients, neuropsychological assessment may play an increasingly important role as the clinician often has less direct access to information typically acquired from teachers or family members when assessing younger children. Neuropsychological assessment of the young adult may serve several purposes: help to support a proper diagnosis by identifying clear-cut attentional or EF deficits, especially for patients who self-report ADHD symptoms but for whom no other data is available; determine changes over time and evaluate treatment effects on cognition; identify young adults who require accommodations in college, such as additional time to complete examinations, in order to compensate for cognitive dysfunction associated with ADHD; and clarify whether a LD is present. Finally, neuropsychological assessment may help both the patient and those close to him or her (e.g., parents, spouse etc.) achieve a better understanding of the patient's individual strengths and weaknesses. This can lead to more realistic expectations of what the person with ADHD can achieve.

12. Summary

ADHD is defined by behavioral characteristics similar to neuropsychological disorders of executive dysfunction. This paper reviews the literature of the neurocognitive characteristics of ADHD from early childhood through adulthood. The group data clearly supports the hypothesis that executive dysfunctions are correlates of ADHD regardless of gender and age, and these EF deficits are exacerbated by co-morbidity with learning disabilities such as dyslexia. Persons with ADHD have improved cognition in certain areas (such as vigilance) when treated by stimulants. However, there is limited data on children under the age of 6, teenagers from 13 to 18 years of age, and adults with ADHD over the age of 40. Studies of individual classification of people with ADHD compared to healthy, non-psychiatric controls do not support the use of neuropsychological tests for the clinical diagnosis of ADHD, and indicate that not all persons with ADHD have EF deficits. Moreover, there is insufficient data comparing the neuropsychological profiles of persons with ADHD vs. other neuropsychiatric disorders. Some persons with ADHD may have deficits in brain reward systems that are relatively independent of EF impairments. Future research should clarify the multiple sources of ADHD impairments, and continue to refine neuropsychological tools optimized for assessment. Finally, it is important for research to clarify whether there is a single, core deficit, such as inhibition, or multiple deficits.

13. Future directions for research

Although there is growing information that identifies neuropsychological abnormalities in ADHD in childhood through adulthood, many questions remain. First, there is still relatively little systematic neuropsychological information on ADHD throughout life, particularly in children <age 6, teenagers, and in adults over the age of 40. Second, most of the research is cross-sectional. It would be very important to evaluate a child sample longitudinally to determine whether the neuropsychological abnormalities change throughout life. Third, combining neuropsychological, structural, and functional MRI measures will allow an evaluation of structure–function relationships in ADHD. Fourth, there is a need for studies to evaluate the increasing evidence of genetic anomalies with measures of brain dysfunction. Although it is premature to identify an association between gene variants and brain abnormalities in ADHD, we believe that when ADHD susceptibility genes have been discovered and confirmed, DNA-imaging resources will provide a useful means of testing hypotheses about gene–brain associations.

An additional important issue in evaluating the significance of neuropsychological deficits in ADHD is whether they are specific to the disorder. Given the emerging pathophysiology of the disorder, involving a widely distributed neural network including prefrontal cortex, anterior cingulate, caudate and possibly other basal ganglia structures, components of the corpus callosum, and the cerebellum, it is possible that the disorder would overlap phenotypically with other disorders in adulthood (e.g., mood disorders) that have dysfunctions in some of those regions. Although some measures have been shown to be specific in childhood comparisons with other neurodevelopmental disorders (Sergeant et al., 2002), it has yet to be shown that the overall profile of neuropsychological functioning is distinct from other disorders.

Another important theoretical question is how best to explain the clinical and neuropsychological picture associated with ADHD. As we noted earlier, a disturbance of attention and EFs has been the dominant model for explaining ADHD over the past two to three decades. However, this model has received only partial support (Sergeant et al., 2003). Sergeant et al. (2003) have recently reviewed the strengths and weaknesses of what they consider to be the five models of ADHD. These include: executive function model, the delay-aversion model, the behavioral-inhibition/activation model, the inhibition model, and the cognitive-energetic model. While it is beyond the scope of this paper to review these models, the field will advance by integrating these models into a broad research agenda to find the best explanatory power for understanding ADHD. For example, Sonuga-Barke has proposed a dual pathway model of ADHD development in which one pathway involves executive deficits associated with abnormalities in frontodorsal striatal circuits, and the other pathway involves delay aversion associated with abnormalities in frontoventral striatal circuits (Sonuga-Barke, 2005). A consensus appears to be growing, based on the empirical data, that the EF, “single deficit” model is insufficient to explain ADHD (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Pennington, 2005; Sonuga-Barke, 2005). These authors suggest that a “new neuropsychological model of ADHD” is likely to encompass subtypes and multiple deficits that include EF deficits, motivational or reward abnormalities, and possibly others (Pennington, 2005). This author shares this conclusion based on the literature reviewed herein. New studies utilizing measures from multiple domains need to be evaluated.

In summary, clinical understanding of the neuropsychology of ADHD needs to be taken into account to provide a greater opportunity for improved and more integrated treatment approaches. For example, an increased knowledge of cognitive difficulties in ADHD will inform treatment providers of ways to incorporate complementary psychopharmacological and/or psychosocial interventions. It will also direct the development of better assessment protocols that might provide a greater rate of both sensitivity and specificity in diagnosing ADHD. Moreover, because ADHD is known to be a heterogeneous disorder with substantial psychiatric and cognitive comorbidity, and because considerable controversy has existed about the nature and validity of ADHD, this article will aid clinicians in developing a better framework for understanding their patients. This greater knowledge of the neuropsychology of ADHD is necessary to help clarify the neurodevelopmental evolution of the disorder, treatment response, and the meaning of the disorder to patients, families, and treating clinicians.

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